because of poor eyesight, arthritis, incoordination, tremor, and fear of the bottle touching the eye. The Autosqueeze, Opticare, and Autodrop, shown in the box, may help in squeezing the bottle or positioning the drop.[4,5] These aids are not available on prescription and have to be purchased; no studies have satisfactorily compared their ease of use and efficacy.

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Passive smoking and health: should we believe Philip Morris's "experts"?

George Davey Smith, Andrew N Phillips

See p 907

A series of adverts has recently appeared in newspapers across Europe comparing the risk of lung cancer from passive smoking with a variety of other apparent risks from everyday activities (see fig 1). The implication is that the increased risk of lung cancer among those exposed to other people's tobacco smoke, of around 20%, is minuscule in comparison with the apparent 500% increased risk of lung cancer associated with a diet high in saturated fat, the 180% increase with frequent cooking with rape seed oil, the 60% increase with drinking 1-2 glasses of whole milk per day, or the 70% reduction in risk associated with high fruit diet. The advert, entitled "What risks do you take?" is cleverly tailored to the public's scepticism about the apparent health risks of everyday activities.[1] A few weeks after the adverts appeared the main headline in a major British Sunday newspaper was "Beefburgers linked to cancer."[2] The frequent appearance of such news stories, which are then often contradicted or reversed by subsequent reports, leads to distrust in pronouncements from experts—what may be called the "now they're saying" syndrome.

The central message of the advert is that passive smoking is not "really a meaningful health risk to people who have chosen not to smoke." Readers were asked to write for a copy of a report "Environmental tobacco smoke and lung cancer: an evaluation of the risk," from a team of authors referred to as "The European Working Group on Environmental Tobacco Smoke and Lung Cancer."[3] The report focused only on the risk of lung cancer, while the adverts referred to an absence of risks of health problems in general with passive smoking. The title of the working group has the ring of authority to it, although unsurprisingly it turns out to be an industry funded enterprise.

Passive smoking and lung cancer: the European working group report

The review of the evidence linking passive smoking to lung cancer risk produced by the tobacco industry sponsored working group is of limited use as a scientific document. It is, however, interesting as a demonstration of the rhetorical mode adopted to dismiss the possibility of a link between passive smoking and lung cancer. The core of the report relates to a summary and meta-analysis of observational epidemiological studies regarding the risk of lung cancer in people who have never smoked but who are exposed to environmental tobacco smoke. These investigations have looked at never-smoking spouses (or cohabitees) of smoking partners, never-smoking people whose parents smoked, and never-smokers exposed to smoke at their workplaces. As with other meta-analyses of this issue,[4,5] an increased risk of about 20% was seen in the spousal studies. The working group considers only studies with female never-smokers with male smoking spouses, on the grounds that there are only nine studies with male never-smokers. This is odd as later the group goes on to carry out subgroup meta-analyses of as few as four individual studies. The suspicion is aroused that the male studies are ignored because they show larger increased risks on average than the female studies.

Having found a significantly increased risk in its meta-analysis of spousal studies, the working group goes on to attempt to discredit this finding. This could be seen as a welcome antidote to the usual practice of scientists attempting to shore up their embattled hypotheses against challenges from newly accruing empirical data. More plausibly, however, it could be seen as a way of getting the accumulated data to fit with the already agreed conclusion. A series of possible biases are considered, which will here be dealt with in turn.

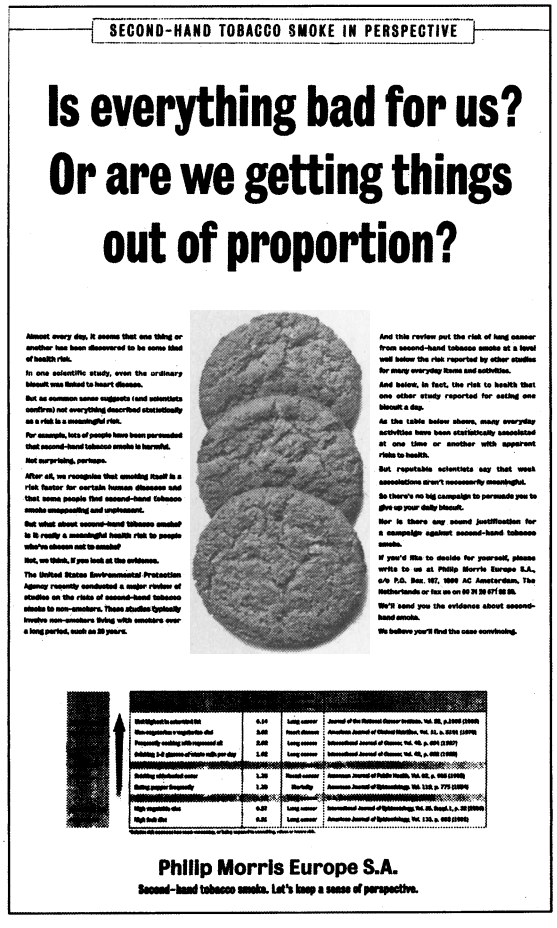

Fig 1—*One of the Philip Morris advertisements*

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BMJ 1996;**313**:929–33

Misclassification

The main criticism of studies showing an increased risk of lung cancer among never-smokers exposed to environmental tobacco smoke is that some current or former smokers may claim to be never-smokers yet be at increased risk of lung cancer because of their previous smoking. If this misclassification of never-smokers is associated with spousal smoking, then it could generate an apparent, but spurious, increased lung cancer risk among passive smokers. Since smokers are more likely to marry other smokers it is plausible that such misclassification is differential—that is, more declared never-smokers married to smokers are actually ex-smokers or current smokers than is the case with declared never smokers married to non-smokers. This issue has been much discussed by Peter Lee.[6-9] Lee is the main authority referred to by the working group, which accepts all his propositions and applies his misclassification model to their data. Nowhere do they point out that Lee is an enthusiastic recipient of tobacco industry financial support and someone who, like the working group, has presented models which are most favourable to the tobacco industry case.[11]

The glaring omission in Lee's model[9] and in the working group report is consideration of the underestimation of the association between exposure to environmental tobacco smoke and lung cancer risk which will be generated by the use of spousal smoking as an indicator of exposure. Lee considers only whether the smoking status of the never-smoking spouses enrolled in studies could be misclassified and states that this has little effect and "will be ignored hereafter."[9] What this fails to acknowledge is that the factor which may cause lung cancer in never-smokers is the inhalation of environmental tobacco smoke, not simply marriage to a smoker. As an indicator of the amount of environmental tobacco smoke that gets into people's lungs, the smoking status of spouses is a highly approximate measure. The ways in which spouses smoke—for example, do they smoke in the house or outside the house only; do they smoke in the same room as other household members; do they have smoking friends who are frequently around the house?—will influence the amount of tobacco smoke the non-smoking spouse inhales. The non-smoking spouses will also be exposed to environmental tobacco smoke from sources outside their homes and unrelated to the smoking status of their partners. The strength of the association between inhaled environmental tobacco smoke and lung cancer will be underestimated when it is indexed by the association between the proxy exposure measure of spousal smoking status and lung cancer. Since spousal smoking history is a fairly distant proxy measure of the amount of environmental tobacco smoke a person inhales this attenuation will be considerable.[10 11]

Cigarette advertisement 1953

Using Lee's method the working group claims that the association in spousal studies it sees in its meta-analysis, of 1.16 (95% confidence interval 1.08 to 1.25) is reduced to 1.08 (1.00 to 1.16), which they suggest is still an overestimate since in some countries greater spousal misclassification occurs, particularly in "traditional countries of Europe such as Greece," where "culture...frowns on female smoking." If, however, they took into account the misclassification of exposure to smoke when the proxy measure of spousal smoking is used, the association would be found to be considerably larger than the estimate from the meta-analysis.

Many problems exist in adjusting for measurement error in epidemiological studies.[10-12] Researchers have commonly applied correction from misclassification to the associations they are interested in—hence increasing them[13]—while ignoring any biases which may have led to overestimation of the exposure-disease associations.[13] In the present case, however, the working group members reversed the usual procedure, so that any association between passive smoking and lung cancer risk becomes less apparent.

Diet as a confounding factor

The working group makes much of the possible confounding of the passive smoking-lung cancer association by dietary factors. They quote the inverse association between intake of fruits and vegetables and lung cancer in many epidemiological studies, which they consider to be due to these food items being rich in antioxidants "for which an efficient cancer protective effect has been hypothesised." They suggest that vitamin E and carotenoids, such as β carotene, are particularly important agents here and quote "LeMarchand *et al* as stating that 'complete control of the confounding effect of β carotene intake is essential in assessing the true magnitude of the passive-smoking/lung-cancer association.'"[14] They then go on to quote evidence that non-smokers living with smokers have lower intakes of these antioxidants and that such confounding may account for the apparent association between environmental tobacco smoke and lung cancer.

Again, what is remarkable about this account is what it leaves out. Firstly, it ignores the fact that the apparent associations between dietary intake and lung cancer could themselves be caused by incomplete control of confounding by smoking. Secondly, the working group fails to point out that virtually all of the studies of diet and lung cancer relate to lung cancers occurring among smokers, lung cancer being very rare among true never-smokers. Thus the effect of diet, at most, is one which potentiates the effect of cigarette smoke. Thirdly, several randomised controlled trials of long term supplementation with antioxidants have produced very inauspicious results, with increases or no change in lung cancer mortality being seen in the groups given β carotene, vitamin E, or vitamin A supplementation.[15-17] The results of these trials created enormous scientific interest and it seems implausible that the working group did not know of them. By far the most robust evidence relating to the proposed confounding by dietary factors seems to be ignored simply because it does not fit in with attempts to dismiss the association between passive smoking and lung cancer.

Evaluating the role of confounding in epidemiological studies is highly problematic.[10 12] Associations which remain after adjustment for apparent confounders can still be spurious, caused by misclassification of confounders or to the presence of unknown—and thus uncontrollable—confounding factors.[18] The usual situation is that investigators do not adequately consider the degree to which confounding can have produced their findings.[19] but the working group bends over backwards to claim that the positive association they find is due to confounding, ignoring important considerations and evidence in the effort. Evaluation of associations in epidemiological studies requires improved study design, with repeat exposure measurements,[10 13] rather than a partisan attachment to one explanation or another.

Environmental tobacco smoke: can it really be bad for you?

The working group devotes much space to details of the components of environmental tobacco smoke. They suggest that the level of carcinogens is too low to be of concern and that even a heavily exposed passive smoker inhales considerably less than one cigarette per day. The composition of such smoke is, however, different from that of mainstream smoke, with some toxins being at lower concentrations and some at higher ones.[4] The importance of this has been shown with respect to atherosclerosis, another illness associated with environmental tobacco smoke.

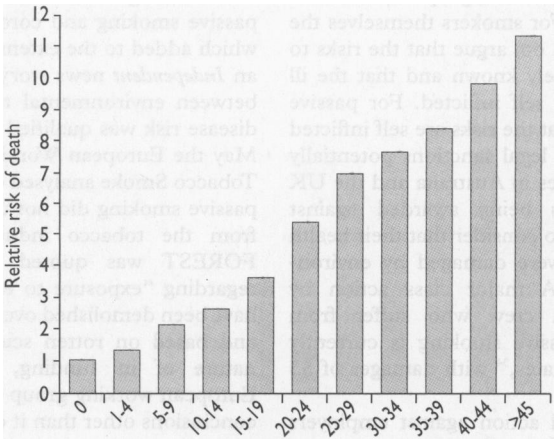

Fig 2—*Relative risk of death from lung cancer according to number of cigarettes smoked per day*

Penn *et al* performed studies in cockerels, supported by the tobacco industry funded Centre for Indoor Air Research. They found that exposure for six hours a day for 16 weeks led to a worsening of the state of atherosclerotic plaques.[20] The tobacco industry claimed the exposure levels were "unrealistically high"[21] and the experiment was repeated at lower exposure levels, with identical results.[22] The investigators monitored environmental tobacco smoke in bars and restaurants and showed that levels which were associated with accelerated atherosclerosis in cockerels were seen in such places. Following the second study the tobacco industry funded body refused to continue supporting this group.[21]

Publication bias

The working group echoes the oft made claim that the apparent association between passive smoking and lung cancer is due to failure to publish negative findings. Such biases can influence meta-analyses,[23] but an exhaustive investigation of whether this occurs with regard to the health effects of environmental tobacco smoke shows that it is not the case.[24]

Smoke gets in your eyes—and lungs

Are the increased risks plausible?

The implication in the working group's conclusions is that the 20% increased risk of lung cancer in passive smokers is not plausible. It is, however, consistent with findings from epidemiological studies of active smoking. Relative risks of death from lung cancer according to number of cigarettes smoked are presented in figure 2.[25] (James Neaton, personal communication.) In this study of a third of a million men a 40% increased risk is seen in those smoking only 1-4 cigarettes per day, compared with those smoking no cigarettes. This increase in risk is underestimated because the no smoking group contains ex-smokers, whose lung cancer risk is raised by their previous smoking. A 20% increased risk of lung cancer in passive smokers compared to never-smokers is fully compatible with the findings of such studies. A necropsy study showed an increased prevalence of epithelial, possibly precancerous, lesions in women married to smokers compared to women married to non-smokers,[26] and tobacco related carcinogens have been identified in the urine of non-smokers exposed to tobacco smoke.[27] In this latter experimental study confounding and misclassification are not issues.

Thresholds and hormesis: is a little bit of exposure good for you?

The working group engages with the literature on thresholds—that is, the question whether there is a level of exposure below which substances that are carcinogenic at high concentrations may not be dangerous. This is a complex and much debated issue.[28] As shown above, there is no suggestion of such a threshold in relation to the association between cigarette smoking and lung cancer. The working group also makes much of a phenomenon referred to as hormesis: the suggestion that low level exposures are beneficial because they stimulate bodily defences and thus reduce the risk of cancer.[28] This argument has been advanced by the nuclear industry with respect to low dose radiation exposure.

The working group approvingly quotes the suggestion that cancer mortality is decreased by exposure to low dose radiation and that "this scientifically-proven benefit invalidates imagined risks from erroneous interpolations of low-dose radiation." This implication—that low level radiation and perhaps a small dose of environmental tobacco smoke are beneficial (with no evidence at all given to support the latter proposition)—is an addition to the attempts, often made by interested parties, to dismiss concerns about low level exposures.[28 29]

Passive smoking and other risks to health

The tobacco industry clearly wants the opinion of the working group that environmental tobacco smoke does

not increase lung cancer risk to be generalised into a general clean bill of health for environmental tobacco smoke. There are, however, many other potential health effects of inhaling smoke. The reduced birth weight and higher risk of perinatal and sudden infant death among the offspring of smokers can be considered the earliest effects of passive smoking.[30 31] In later childhood there is evidence of poorer respiratory health and increased risk of asthma, middle ear infections, and sore throats resulting from exposure to environmental tobacco smoke.[30 32] In adults the risk of chronic obstructive airways disease is higher among those exposed to environmental tobacco smoke and lung function is worse.[33] A considerable body of epidemiological and experimental data suggests that ischaemic heart disease risk be increased by exposure to environmental tobacco smoke.[21] The tobacco industry and its friends clearly hope that doubts cast on the association between such smoke and the risk of lung cancer will influence considerations regarding the effects of environmental tobacco smoke on other forms of ill health.

Passive smoking: why is the tobacco industry so concerned?

The tobacco industry is clearly going to considerable trouble to discredit evidence that environmental tobacco smoke is detrimental to health. Two main reasons for this can be discerned. Firstly, the threat of legal cases resulting in compensation for those whose health has been damaged by passive smoking represents a considerable economic threat. For smokers themselves the tobacco companies can, and do, argue that the risks to health have been made widely known and that the ill effects are at least partially self inflicted. For passive smokers, however, claims that the risks are self inflicted are less sustainable and the legal sanctions potentially more damaging. Several cases in Australia and the UK have resulted in damages being awarded against employers to individuals who consider that their health or employment prospects were damaged by environmental tobacco smoke.[34] A major class action by non-smoking airline cabin crew who suffer from illnesses attributable to passive smoking is currently under way in the United States,[34] with damages of $5 billion being claimed.

Secondly, threats of legal action against employers who allow their employees to be exposed to others' smoke will lead to the imposition of stringent smoking restrictions at workplaces, which will in turn reduce cigarette sales.[35]

How the tobacco industry defends your right to smoke

Considerable resources are devoted to funding apparent "experts" to cast doubt on the health damaging effects of smoking. The working group is a typical example. A Medline search on the working group's members revealed no evidence of expertise in epidemiology; indeed the industry rarely funds epidemiologists.[28] Thus a review which focused largely on epidemiological findings was carried out by a group whose relevant expertise is in doubt.[36]

Professor J R Idle, chairman of the working group, thanks his team "for their tireless efforts in otherwise extremely busy agendas. The integration of such varied expertise into a cohesive and unified statement is a testimony to the dedication and both personal and professional qualities of the working group."[3] These "tireless efforts" were presumably encouraged by the funding which was received for this undertaking. As we saw above, when researchers funded by the industry come to conclusions counter to their sponsors' needs, such funding can be removed. The expenses of the working group are tiny compared with the potential loss of revenue through reduced cigarette sales and potential legal fees in litigation, making these experts a very cheap investment indeed.

> **"Doubt is our product"**
>
> The main tactic of the tobacco industry is to promote the impression that there is considerable uncertainty and scientific controversy about the damaging effects on health of smoking. In 1969 an internal industry document read:
>
> Doubt is our product since it is the best means of competing with the 'body of fact' that exists in the mind of the general public. It is also the means of establishing a controversy. If we are successful at establishing a controversy at the public level then there is an opportunity to put forward the real facts about smoking and health."[28]
>
> In 1978 a report to the US Tobacco Institute stated that public worries about smoking were "the most dangerous development to the long term viability of the tobacco industry that has yet occurred" and that the strategic and long run antidote to the passive smoking issue is ... developing and widely publicising clear-cut, credible medical evidence that passive smoking is not harmful to the non-smoker's health."[40]

The way such investments can pay off is shown by news coverage of a recent study showing a link between passive smoking and coronary heart disease,[37] a study which added to the extensive evidence in this area.[21] In an *Independent* news story a report of the positive link between environmental tobacco smoke and coronary disease risk was qualified with the statement that "last May the European Working Group on Environmental Tobacco Smoke analysed 48 studies and concluded that passive smoking did not cause cancer."[38] A spokesman from the tobacco industry sponsored organisation FOREST was quoted as saying that the claims regarding "exposure to environmental tobacco smoke have been demolished over the years, shown to be bogus and based on rotten science." The fact that, by the nature of its funding, the authoritative sounding European working group was unlikely to come up with conclusions other than it did is lost in such reporting.

Conclusion

Investigating the links between exposure to environmental tobacco smoke and disease is beset with methodological problems, while the public response to reports of new health risks is understandably a sceptical one. The tobacco industry capitalises on this situation to protect its commercial interests, through the promotion and magnification of confusion. The industry is guarded about its real knowledge on the health damaging effects of tobacco smoke[39] and tries to influence opinion through the selective funding of research, support of the publication of pro-industry opinions, and intimidation of its opponents. The partial and biased nature of the adverts and "expert" report at the heart of the latest industry campaign represents a continuation of its characteristic behaviour.

We thank Dr James Neaton for providing the updated analyses of lung cancer mortality according to smoking status from the MRFIT screening study and Anne Rennie for help in preparing the manuscript.

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A memorable patient

Facts and figures

During their medical education doctors are taught to translate the symptoms of the patients into objective parameters in order to come to the right diagnosis. Teachers also remind their students that it is not always possible to express the facts into figures, and a good doctor should never refrain from watching and listening to the patient. In medical literature, however, figures seem to be all that matter, and it is easy to forget the old lesson.

After I had finished my training in pulmonology I became one of the doctors responsible for a new lung transplantation programme. Many things had to be learnt along the way. One of the things that had to be evaluated was the choice of transplantation procedure for different diseases. Patients with pulmonary emphysema were initially treated by bilateral lung transplantation, and Mr A was the first patient with emphysema to be scheduled for a single lung transplantation. He was a friendly and optimistic middleaged man, raised in a no nonsense harbour town where working was more appreciated than talking.

After Mr A became convinced that a single lung transplantation was as good an option as a bilateral lung transplantation, he was looking forward to the operation and the expected possibilities afterwards. Unfortunately, the facts were completely different. After his lung transplantation he was confronted with repeated periods of rejection and suffered a persistent primary cytomegalovirus pneumonitis. Subsequently he developed an aspergilloma in his native lung and he had many subjective complaints during his treatment with liposomal amphotericin B. After this episode a paramediastinal and cavitating lesion developed in his transplanted lung. Several wearisome bronchoscopies could not establish a diagnosis.

Mr A took all the misfortune with admirable optimism and perseverance, but with progressive dyspnoea his hope started to fade away. He did not tell us directly, but it was more in the words he did not say. One night, during a visit to a theatre, my bleeper went off asking me to go the hospital to see Mr A. While cycling to the hospital I realised that it was unusual for this unpretentious man to ask for a doctor at this time of the day. In his scantily lighted room I found him in respiratory distress, sitting upright in the middle of his bed. We did not say much, and after a few minutes his dyspnoea decreased somewhat. He looked at me with tear filled eyes and with sadness said, "I know I won't get better, but thanks for everything."

Soon after that Mr A went home. He did not want to come to the hospital any more. He died 371 days after his transplantation due to extensive post-transplant lymphoproliferative disease.

Many people are delighted with the one year survival rate that we achieved with the lung transplantation programme, especially as it is slightly better than most figures published in the literature. In fact, Mr A was one of the contributors to our excellent one year survival rate. He made me realise again that figures tell you only some of the facts.—GREGOR MANNES *is a pulmonologist in the Hague, the Netherlands*

We welcome filler articles of up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake,* or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk.